Review Article

Lean meat and heart health

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The general health message to the public about meat consumption is both confusing and misleading. It is stated that meat is not good for health because meat is rich in fat and cholesterol and high intakes are associated with increased blood cholesterol levels and coronary heart disease (CHD). This paper reviewed 54 studies from the literature in relation to red meat consumption and CHD risk factors. Substantial evidence from recent studies shows that lean red meat trimmed of visible fat does not raise total blood cholesterol and LDL-cholesterol levels. Dietary intake of total and saturated fat mainly comes from fast foods, snack foods, oils, spreads, other processed foods and the visible fat of meat, rather than lean meat. In fact, lean red meat is low in saturated fat, and if consumed in a diet low in SFA is associated with reductions in LDL-cholesterol in both healthy and hypercholesterolemia subjects. Lean red meat consumption has no effect on *in vivo* and *ex vivo* production of thromboxane and prostacyclin or the activity of haemostatic factors. Lean red meat is also a good source of protein, omega-3 fatty acids, vitamin B_{12}, niacin, zinc and iron. In conclusion, lean red meat, trimmed of visible fat, which is consumed in a diet low in saturated fat does not increase cardiovascular risk factors (plasma cholesterol levels or thrombotic risk factors).

Key Words: nuts, meat, heart disease, CHD risk factors, LDL cholesterol, saturated fat, polyunsaturated fatty acids.

Introduction

A balanced and broadly varied dietary intake plays a critical role in human health. Human and pre-human diet history shows that for a period of at least two million years the human ancestral line had been consuming increasing quantities of meat. During that time, evolutional selection was in action, adapting our genetic make up and hence our physiological features to a diet high in lean meat.[1] This meat was wild game meat, low in total and saturated fat (SFA) and relatively rich in polyunsaturated fatty acids (PUFA).[2]

Since 1984, there have been a number of epidemiological and prospective studies that have suggested that red meat consumption is associated with increased risk of coronary heart disease (CHD).[3-5] Furthermore, vegetarians living in industrialised societies such as the Seventh Day Adventists have a low incidence of CHD.[3] In addition, the individual SFA intakes (lauric, myristic, palmitic and stearic acid) showed a high correlation with mortality from CHD in the Nurses' Health Study[6] and the Seven Countries Study.[7] In the Nurses' Health Study from USA, the dietary intake of stearic acid, with beef as a primary source (as main dish, mixed dish, sandwich and hamburgers) was reported to increase the risk of CHD even more than did myristic, palmitic and lauric acids.[6] Cross sectional studies have shown that omnivores had a significantly higher intake of total fat and SFA which was associated with increased plasma/serum total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) compared with vegetarians.[8,9] After further analysis of the fat sources, based on food frequency questionaries, it was found that only 8% of daily fat intake was gained from meat cuts, and 12% from meat dishes and meat products. Hidden fats in fast foods, snack foods and other processed food were the primary sources of dietary SFA intake.[10]

It is well known that meat fat from all animals is rich in SFA, and in order to promote reductions in dietary total and SFA intakes, many health professionals have encouraged people to reduce their consumption of meat, a message which may have contributed to the decline in meat consumption over the past three decades.[11] For example, replacing red meat with white meat might have benefits in relation to lipoprotein lipids, since red meat was regarded to be higher in SFA and lower in PUFA compared with white meat.[12] There is confusion amongst consumers about the impact of lean meat and visible fat on human health, because it is commonly, but incorrectly thought that lean meat will have the same SFA content as the visible fat of meat.

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This review is based on publications in the international literature, and highlights that lean red meat is low in SFA and that consumption of lean red meat does not increase plasma/serum cholesterol levels or increase thrombotic risk. In this review, red meat is defined as beef, veal and lamb and lean red meat is based on Heart Foundation guidelines (<10% total fat).[13]

Is lean red meat rich in saturated fat?

As shown in Table 1, the SFA content of lean red meat is less than 1.5g/100g lean red meat compared with more than 37g SFA/100g of visible fat from red meat. The main lipid in lean meat tissue is phospholipid (PL) which makes up the major portion of the cell membrane structure, and these are rich in PUFA, while the other main lipid in lean meat is triacylglycerol (TAG) which in beef and lamb are enriched in SFA and monounsaturated fatty acids (MUFA).[14] The PL concentration in lean red meat is relatively constant and independent of the lipid level whilst the level of TAG increases as the total lipid level increases.[15] Therefore, as the total lipid content of the lean red meat increases there is an increase in the SFA and MUFA content. In other words, the fatty acid composition of meat depends on the total lipid content of the meat.[14-17]

Visible fat from ruminant meats (beef and lamb) has a higher lipid level (>80%) than that from pork and poultry (<80%).[18] Once visible fat is trimmed, lean red meat often contains less than 5% lipid, as there is usually very little marbling within the meat in Australia. The lipid level in lean meat ranges from 1% in turkey to 4.2% in lamb meat.[18-20] Lean meat is relatively higher in PUFA and lower in SFA compared with visible fat of meat. As Table 1 shows, lean meats all contain less than 2g SFA/100g, which is similar to the SFA content of milk.[18]

Does a diet rich in lean red meat increase plasma/serum cholesterol levels?

(a) Studies in beef. In response to the confusion about lean meat and visible fat of red meat and its link with hyperlipidemia, a 5 week dietary intervention study was conducted, which aimed to differentiate between the effects on plasma cholesterol of lean red meat (beef) and meat fat (beef dripping). Ten (5 male, 5 female) healthy subjects aged 22 to 30 years (BMI = 23.8 ± 1.0) participated in the study. In the first week the subjects consumed their usual diet, which included 30 to 100g raw beef/day (38% energy as fat, PUFA:SFA= 0.27).[21] In the second and third weeks, the subjects consumed a very low fat diet containing 500g/day of very lean beef (<10% energy as fat, PUFA: SFA 0.49).[20] This amount of lean red meat is more than double the mean intake of red meat by Australians.[9] Results showed there was a 17% and 27% reduction in total and LDL-C levels, respectively, after two weeks of a very low fat, lean beef diet.[21] When beef fat (dripping) was added back to the diet in a step-wise fashion over the final two weeks of the study (29% energy as fat, PUFA: SFA=0.21), the beneficial effects on the total and LDL-C levels were reversed. HDL-C was not affected by any of these dietary changes.[21] This result clearly indicated that visible fat of meat, rather than the lean red meat increased serum cholesterol concentration.

In subsequent studies, the effect of the inclusion of lean beef on plasma lipoprotein lipid levels in people with normal serum and lipoprotein lipids, and with hypercholesterolemia was investigated.[22,23] In the first study, the effects on serum cholesterol levels of a diet rich in lean beef, with the addition of other fats - olive oil and safflower oil in people with normal serum and lipoprotein lipids was studied.[22] These oils were chosen because both have beneficial effects on plasma cholesterol levels. Twenty-two (11 male, 11 female) healthy free-living subjects aged 30 to 55 years participated in the study. Subjects were assigned to either the safflower oil group (6 male, 5 female) or the olive oil group (5 male, 6 female). In the first week the subjects ate their usual diet; in the second and third weeks all subjects ate a very-low-fat diet (9% of energy, SFA 3.7% energy) rich in lean beef (500 g/day). In the fourth and fifth weeks the subjects continued to consume the lean beef daily, and the fat content of the diet was increased in a stepwise fashion to 20% and 30% of energy, respectively, by substituting safflower oil or olive oil for carbohydrate. The LDL-C concentrations decreased significantly by 13% to 14%, and HDL-C concentrations decreased by 20% to 25% in subjects after 2 weeks on the very low fat, lean beef diet. The LDL-C concentrations remained low after the addition of safflower oil or olive oil to the very-low-fat diet rich in lean red meat. In the second study,[23] twenty-four (14 male, 10 female) free-living subjects, aged from 22 to 66 years with mildly elevated serum cholesterol concentrations participated during consecutive 8-week periods. In the first two weeks all subjects consumed their usual diet, which contained an average of 154g of cooked meat/day (beef, veal, lamb, pork, chicken). In the following six weeks, subjects were randomised to start with one of the two diets: the modest fat diet (30% energy as fat) containing olive oil, or the very low fat diet (10% energy as fat). Both diets contained ~300 g/day of fat-trimmed lean beef (raw weight). Serum concentrations of TC and LDL-C decreased significantly by 11% and 13%, respectively, on the modest fat diet, and by 14% and 13% on the very low fat diet, respectively. However, there were no significant differences between the two diets on serum TC and

Table 1. Fatty acid profiles of lean and visible fat of meats (mg/100g)

	Lean meat			Visible fat		
	SFA	MUFA	PUFA	SFA	MUFA	PUFA
Beef	384 ± 56	393 ± 89	151 ± 19	37403 ± 5244	32167 ± 5062	1345 ± 90
Lamb	1485 ± 247	1443 ± 335	333 ± 92	39214 ± 3941	25659 ± 1438	2420 ± 210
Chicken	571 ± 152	812 ± 220	362 ± 129	19987 ± 1274	33786 ± 1259	8384 ± 2022
Pork	544 ± 90	651 ± 165	305 ± 18	22688 ± 4109	28055 ± 3325	11523 ± 3107
Duck	366 ± 108	339 ± 69	333 ± 88	22197 ± 1908	39765 ± 4699	9828 ± 1405
Turkey	292 ± 83	240 ± 149	262 ± 46	13779 ± 5518	17946 ± 7591	8144 ± 3459

LDL-C concentrations. The HDL-C concentrations were significantly reduced for both diets, but the LDL-C: HDL-C ratio was significantly lower in the modest fat diet compared with the very low fat diet.[23] These results indicate diets rich in fat-trimmed lean beef and low in SFA (not total fat) were effective in reducing plasma/serum TC and LDL-C levels. The SFA content of the two dietary regimes were 4.2 and 6.7% of energy compared with the subjects usual diet SFA intake of 15.1% energy.

(b) Studies using mixed meat (chicken, beef and pork): Effect of mixed meat and meat products with different amounts of animal fat on plasma and lipoprotein lipids have been investigated in 15 free-living hyperlipidaemic men with a mean age of 49.9 years.[23] The study comprised a 4-week reference period (diet A, typical of the average British diet, 42% energy from fat with 21% SFA), followed by two four-week experimental dietary periods (diet B, 35% energy from fat with 14% SFA, and diet C, 27% energy from fat with 8% SFA with a fibre supplement). Daily consumption of meat and meat products was the same in each experimental dietary period (180 g/day, 20g cooked and 160g raw weight). In this study the mixture of meat and meat products included carcase meat, offal, bacon cooked meats, canned meats, sausages, meat pies and other meat products. The fat content of meat and meat products was 16% for diet A, and 8.5% for diets B and diet C. Compared with diet A, the plasma concentrations of TC and LDL-C fell by 8.6% and 11% in diet B, by 18.5% and 23.8% in diet C. TAG, HDL-C and BMI did not change appreciably during the study.[23] Based on these results the authors suggested that it was possible to reduce plasma cholesterol levels by reducing dietary fat (animal fat) rather than removing meat from the diet.

(c) Comparison of red meat and white meat: In a recent crossover study, 18 hypercholesterolemic men aged 21–73y were assigned to either a lean beef or lean fish or poultry diet with 30% of energy from fat, for at least 26 days each with a 6 week washout period. The three experimental diets reduced plasma TC and LDL-C by 5-9% compared with baseline. No significant differences between the 3 experimental diets were found in these lipid variables.[25] In a 13-week dietary intervention study, 38 free-living hypercholesterolemic men completed two test diets for 5 weeks each. The test diets contained either 85g of cooked beef (8% fat) or 85g of cooked chicken (7% fat) with 7% to 8% of energy from SFA. All food was supplied during the study period. Plasma concentrations of TC and LDL-C were significantly reduced by 7.6% and 9% on the beef diet, by 10.2% and 11% on the chicken diet, respectively. There were no significant differences between the beef and chicken diets on concentrations of plasma TC, HDL-C, TAG, and LDL-C. The plasma TAG concentrations did not change for either test diet groups.[26] In another study, 191 hypercholesterolemic subjects (107 male, 84 female) were instructed to consume at least 80% of their meat in the form of lean red meat or lean white meat. Fasting serum lipid levels were assessed at 4, 12, 20, 28, and 36 weeks after commencement of the study. There were no significant differences between lean red meat and lean white meat phases on serum concentrations of TC, LDL-C, HDL-C and TAG at the end of 36 weeks randomisation. Compared with baseline, both the lean red meat and lean white meat diet groups resulted in a nearly identical reduction in TC by 1% and LDL-C by 2%, and increase in HDL-C by 2%. TAG concentrations remained similar to baseline values.[27] These results suggested that lean red meat (beef, veal, and pork) and lean white meat (chicken and fish) had similar effects on plasma/serum concentrations of TC, LDL-C, HDL-C and TAG.

(d) Lean red meat versus soybean products: It has long been recognised that consumption of soy products reduces blood cholesterol levels.[28,29] The effect of lean red meat versus soybean products on plasma and lipoprotein lipids in healthy and overweight individuals has been compared in two recent studies. In the first study, 36 women, mostly overweight or obese with BMI 32.4 ± 5.2 kg/m^2, aged 30 to 61 years were assigned non-randomly to either the red meat group (N=19) or soybean group (N=17) for 16 weeks. Both diets were equienergetic (1500 kcal) designed to lead to weight loss. In the red meat diet, subjects consumed lean beef 150g/day at least 5 days per week and they consumed either fish or non-soy legumes for the 2 remaining days (23% of energy from fat with 6.4% SFA).[30] In the soybean diet, subjects were encouraged to consume soy protein (130g per day dried soy bean) at least 5 days per week, and chicken, fish and other legumes for the remaining days (22% of energy from fat with 4.2% SFA). There were body weight reductions of 7.8kg and 7.6kg for the lean meat diet and soybean diet, respectively, at the end of the 16 weeks compared with baseline. Compared with baseline, the concentrations of TC were significantly decreased by 15% for the lean meat diet group and 9% for the soybean diet group, LDL-C by 16% and 10%, and TAG by 13% and 18% at the end of the 16 weeks, respectively. In other words, lean meat and soybean diets had a parallel effect on reduction of plasma TC, LDL-C and TAG. The second study was a randomised crossover dietary intervention study, 42 free-living healthy males aged 35-62y participated in the study. One diet contained lean beef (150g/d) and the other diet contained 290 g/d tofu. The two diets were similar in energy, protein, macronutrients and fibre. Study length was one month for each diet and in between diets subjects resumed their usual diets for 2 weeks as a wash-out period. The TC, HDL-C and TAG were significantly lower in the tofu diet compared with the lean beef diet. Compared with baseline, HDL-C was significantly increased and the LDL-C:HDL-C ratio was significantly decreased in the lean beef diet, but there were no significant changes in the tofu diet. There were no significant differences in the LDL-C: HDL-C ratios between the two diets.[31] These results showed that lean beef and soybean-based diets had equal beneficial effects on plasma/serum and lipoprotein lipids, and body weight reduction.

In summary, these studies showed that the visible fat of meat, rather than the lean meat increased serum cholesterol concentrations. Diets rich in fat-trimmed lean beef and low in saturated fat, not total fat, were effective in reducing plasma/serum TC and LDL-C levels. Lean

red meat (beef, veal, and pork) and lean white meat (chicken and fish) had similar effects on plasma/serum concen-trations of TC, LDL-C, HDL-C and TAG. Lean meat (beef) and soybean-based diets had equal beneficial effects on plasma/serum and lipoprotein lipids, and body weight reduction.

Does lean red meat increase thrombotic risk factors?

(a) Background: From an epidemiological point of view, it has become clear that the traditional cardiovascular risk factors such as elevated serum cholesterol, cigarette smoking, elevated blood pressure, and a positive family history of CHD were not adequate to identify many patients at risk for future thrombotic events. However, evidence has accumulated which suggests that specific haemostatic and thrombotic factors exist which may greatly increase an individual's risk for acute thrombosis. In general, these factors include increased aggregability of platelets, increased reactivity of the coagulant system (hypercoagulability), abnormalities of blood rheology and depressed fibrinolytic mechanisms.

Arterial thrombosis is generally recognised to play a major role in the transition from stable to acute ischaemic heart and cerebral diseases, manifested by unstable angina, acute thrombotic infarction and sudden death. Platelet aggregation is an early event in the development of thrombosis. It is initiated by thromboxane A_2 (TXA_2), a potent platelet aggregation agent and vascular contractor, produced from arachidonic acid (AA; 20:4n-6), a long chain n-6 PUFA in the platelet membrane.[32,33] However, AA metabolism to TXA_2 can be down regulated by the 20- and 22-carbon n-3 PUFA found in fish, fish oils and lean red meat. A diet with a high n-6 to n-3 PUFA ratio can cause an increased tissue n-6 to n-3 PUFA ratio (i.e increased AA to EPA ratio), which may promote the production of TXA_2 and therefore increase the possibility of platelet aggregation. All meats are a major source of dietary AA, and this AA may contribute to the pool of tissue AA and thereby to an increased risk of platelet aggregation and thrombosis.[20] On the other hand, lean red meats and fish are also major sources of EPA, DPA, and DHA, which may provide a protective effect against platelet aggregation.

(b) Cross sectional study: A cross sectional study showed that the n-3 PUFA in plasma and platelet PL was significantly lower in both lacto-ovo vegetarian and vegan groups compared with meat-eaters in the study population.[9] Collagen and ADP stimulated *ex vivo* whole blood platelet aggregation showed a significant opposite trend to both plasma and platelet PL n-3 PUFA. Mean platelet volume (increased mean platelet volume is an independent risk factor for CVD) and plasma 11-dehydro-thromboxane B_2 (11-dehydro TXB_2), a stable metabolite of thromboxane A_2, were found to be higher in lacto-ovo vegetarian and vegan groups compared with meat-eaters. In other words, the meat eaters had less reactive platelets.

(c) Does lean red meat consumption cause an increased TXA_2 production in humans? It is known that diets rich in red meat increase the plasma level of AA,[34,35] but no studies have established whether this promotes an increase in thrombosis tendency. To answer this question, Mann *et al.*,[36] investigated the effect of diets rich in lean red meat, lean white meat and fish on *ex vivo* platelet aggregation, thromboxane and prostacyclin production in 29 healthy and non-smoking volunteers (14 male, 15 female) aged 22 to 52 years. Each subject completed three, 3-week low-fat (<15% energy as fat) dietary interventions: Atlantic salmon (133±32 g/day/ head), lean beef and lamb (351±104 g/day/head) and lean turkey (231±52 g/day/head). Because the dietary fatty acid composition of subjects' habitual diets was not the same, in each dietary intervention subjects consumed a vegetarian diet for 1 week followed by 2 weeks on the experimental diet. Between each diet period, subjects resumed their usual diet for a 3-week washout period. EPA contents of the experimental diets were 1mg/d in the lean turkey diet, 70mg/d in the lean beef diet and 847 mg/d in the fish diet, while the 20:4n-6 content of each diet (mean 140mg/d) was approximately double the usual intake. Platelet phospholipid (PL) AA was significantly decreased in the fish diet compared with baseline and week 1 vegetarian diet. Compared with week 1 vegetarian diet, platelet PL EPA was significantly increased in the fish and lean beef diets, but significantly decreased in the lean turkey diet. The serum PL ratios of AA/EPA were significantly increased in the lean turkey diet and decreased in the fish and lean beef diets. Neither white nor red meat diets affected collagen- and thrombin-induced platelet aggregation, *ex vivo* platelet TXB_2 production during aggregation, or the systemic PGI_2 or TXA_2 production measured as urinary excretion of 2,3-dinor-6-keto-PGF1 alpha (PGI_2-M) and 11-dehydro-TXB_2 (TXB_2-M). The fish diet significantly decreased platelet TXB_2 production (collagen-stimulated) and urinary excretion of TXA_2-M, while PGI_2-M levels were unaltered. These results indicate that diets which double the usual AA intake using white meat (175-330 g/d) or red meat (275-530 g/d) were not associated with an increased platelet aggregation and TXA_2 production. However, this does not rule out the adverse effects of AA at higher levels in the diet or for more prolonged periods.[36]

(d) Does red meat alter coagulation?

Some early studies in animals suggested that SFA are prothrombotic, and it was suggested that stearic acid might promote thrombosis.[37,38] Furthermore, Factor VII coagulant activity was significantly increased when citrated plasma was incubated *in vitro* in the presence of injected free stearic acid[39] and subsequently it was proposed that dietary fat induced activation of factor VII through effects on the free stearic acid concentration in plasma.[40]

However, human studies showed that stearic acid is not pro-thrombotic. For example, the intake of 20 g/day of stearic acid from shea butter (42% stearic acid) for 3 weeks resulted in a significant reduction in factor VII coagulant activity compared with diets containing palm oil (43% palmitic acid) and palm-kernel oil with high-oleic sunflower oil (10% myristic acid, 30% lauric acid) in 15 healthy young men.[41] A recent randomised cross-over dietary intervention study showed that a stearic acid-enriched diet improved thrombogenic and atherogenic risk factors compared with a palmitic acid-enriched diet

in 13 healthy and non-smoking men aged 35 ± 12 years.[42] Each subject was randomly assigned to either the stearic acid diet which comprised 35% hydrogenated canola oil and 65% monounsaturated sunflower oil (stearic acid intake of 19.4 ± 4.5 g/day /head) or the palmitic acid diet which comprised of 50% palm stearine and 50% monounsaturated sunflower oil (palmitic acid intake of 22.5± 5.3g/day /head) for 4 weeks with a 7 week washout period between the two dietary periods. The diets consisted of approximately 30% of energy as fat. Collagen and ADP-induced *ex vivo* platelet aggregation, mean platelet volume and coagulation factor VII activity were significantly decreased at the end of the stearic acid diet compared with the palmitic acid diet.

Effect of lean red meat (150 g/day/head) and tofu (290 g/day/head) on plasma factor VII activity and fibrinogen concentration have been studied in 45 healthy men aged 35 to 62 years. Subjects were randomly assigned to the lean meat or tofu diet for one month. Subjects consumed their usual diet for two weeks as a "washout period", and then consumed the second diet for another month. There was no significant difference between the lean meat and tofu diet on plasma factor VII activity and fibrinogen concentration. This result indicates that lean red meat does not cause an increase in coagulation factors (plasma factor VII coagulant activity and fibrinogen concentration).[27]

(e) Is lean red meat a significant source of n-3 PUFA?

Lean red meat contains α-linolenic acid (18:3n-3), eicosapentaenoic acid (EPA, 20:5n-3), docosapentaenoic acid (DPA, 22:5n-3) and docosahexaenoic acid (DHA, 22:6n-3), however, the long chain (LC) n-3 PUFA (20 and 22 carbon n-3 PUFA) are not found in visible fat of meats. The predominant LC n-3 PUFA in lean meat is 22:5n-3, with the total LC n-3 PUFA content is approximately 60 mg/100 g for lean beef and 120 mg/100 g for lean lamb.[18]

Effect of ingestion of a diet rich in lean beef on the fatty acid composition of plasma PL have been investigated.[35] Firstly thirty-three healthy free-living volunteers (16 male, 17 female) aged between 22 to 66 years consumed 500g raw lean beef with no added other fat daily for 2 weeks. Plasma PL composition of 20:5n-3 and 22:5n-3 was significantly increased at endpoint compared with usual diet. Then subjects were divided into three dietary groups with same amount of lean beef, but by an increase in the dietary fat by 10% each week for the further two weeks. The added fat was comprised of beef fat, or olive oil or safflower oil. Lean beef plus beef fat and lean beef plus olive oil were associated with significant increases in plasma PL composition of 20:5n-3 and 22:5n-3. In contrast, lean beef plus safflower oil (>75% of 18:2n-6) resulted a significant decrease in 20:5n-3 and increase in 22:5n-3. Dietary 18:2n-6 intake inhibits EPA incorporation from dietary fish-oil supplements and from dietary meat.[44,45] The order of potency in raising platelet PL EPA level was greatest with fish consumption or fish oil supplementation, moderate with lean red meat or linseed oil and least effective with canola oil.[46] The content of serum PL 20:5n-3, 22:5n-3 and 22:6n-3 and platelet PL 20:5n-3 and 22:6n-3 significantly increased after 2 weeks of a lean beef diet (351 ± 104 g per day) and an Atlantic salmon diet (133 ± 52 g per day) in 29 healthy adults (14 men, 15 women, mean age 34.8 years) compared with baseline.[36] These results indicate that lean red meat does improve the n-3 PUFA status in humans, and dietary 18:2n-6 inhibits the incorporation of dietary 20:5n-3. While the amounts of LC n-3 PUFA in lean meat are not as high as in fish, it has been reported that red meat contributes 20% of LC n-3 PUFA in Australian adults based on the 1995 Australian Nutrition Survey.[47]

These studies showed that lean red meat consumption does not cause an increased thrombotic tendency in relation to platelet aggregability, mean platelet volume, eicosanoid biosynthesis, plasma factor VII coagulant activity and fibrinogen concentration. In addition, lean meat raises levels of LC n-3 PUFA in plasma and platelets, fatty acids which have been reported to be have beneficial effects on CVD prevention.

Red meat improves vitamin B_{12} and homocysteine status

Hyperhomocysteinaemia is an important independent risk factor for cardiovascular disease.[48] Homocysteine can be *in vivo* metabolised to cysteine and remethylated to methionine, later involves the enzymatic transfer of a methyl group from 5-methyltetrahydrofolate (5-methyl-THF) to homocysteine with vitamin B_{12} (methylcobalamin) as a coenzyme.[49] Vitamin B_{12} deficiency might cause an accumulation of homocysteine, leading to a hyperhomocysteinaemia. It has been reported that meat consumption was significantly positively correlated with dietary intake of vitamin B_{12} based on 24-hour dietary recalls in a cross sectional study in 504 aged 19 to 28 years omnivores.[50] Serum vitamin B_{12} concentration was significantly lower in vegetarians compared with omnivores,[51,52] because vegetarians, especially vegans can only gain limited vitamin B_{12} from vegetables, soy and yeast. The main dietary sources of vitamin B_{12} include lean meat, seafood, egg yolk, milk and dairy products.[53] In a cross sectional study which involved 18 vegans, 43 lacto-ovo vegetarians, 60 moderated meat eaters (<285 g/day) and 18 high meat eaters, it was found that serum vitamin B_{12} concentration was significantly negatively correlated with plasma homocysteine concentrations.[54] These results showed that dietary meat is a primary source of vitamin B_{12} and is positively associated with plasma vitamin B_{12} concentration and negatively associated with plasma homocysteine levels. Lean meat is also a good source of protein, zinc and iron.[1]

Conclusion

Dietary intervention and cross-sectional studies showed that visible fat trimmed lean red meat does not raise blood cholesterol and LDL-cholesterol levels, and does not change thrombotic risk factors such as thromboxane and prostacyclin production, platelet function and haemostatic factors. In fact, low SFA diets containing lean red meat are associated with a reduction of LDL-cholesterol levels in both subjects with hypercholesterolemia and healthy subjects. Lean red meat is also a good source of protein, omega-3 fatty acids, vitamin B_{12}, niacin, zinc and iron.

References

1. Mann NJ. Dietary lean red meat and human evolution. Eur J Nutr 2000; 39: 71-79.
2. Eaton SB, Eaton SB, Sinclair AJ, Cordain L, Mann NJ. Dietary intake of long-chain polyunsaturated fatty acids during the paleolithic. World Rev Nutr Diet 1998; 83: 12-23.
3. Snowdon DA, Phillips RL, Fraser GE. Meat consumption and fatal ischemic heart disease. Preventive Medicine 1984; 13: 490-500.
4. Mahley RW, Palaoglu KE, Atak Z, Dawson-Pepin J, Langois AM, Cheung V, Onat H, Fulks P, Mahley LL, Vakar F, Ozbayrkci S, Gokdemir O, Winkler W. Turkish Heart Study: lipids, lipoproteins, and apolipoproteins. J Lipid Res 1995; 36: 839-859.
5. Huijbregts PPCW, Feskens EJM, Kromhout D. Dietary Patterns and Cardiovascular Risk Factors in Elderly Men: The Zutphen Elderly Study. Int J Epidemiol 1995; 24: 313-320.
6. Hu FB, Stampfer MJ, Manson JE, Ascherio A, Colditz GA, Speizer FE, Hennekens CH, Willett WC. Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. Am J Clin Nutr 1999; 70: 1001-1008.
7. Kromhout D, Menotti A, Bloemberg B, Aravanis C, Blackburn H, Buzina R, Dontas AS, Fidanza F, Giampaoli S, Jansen A Karvonen M, Katan M, Nissinen A, Nedeljkovic S, Pekkanen J, Pekkarinen M, Punsar S, Rasanen L, Simic B, Toshima H. Dietary saturated and trans fatty acids and cholesterol and 25-year mortality from coronary heart disease: the Seven Countries Study. Prev Med 1995; 24: 308-315.
8. Li D, Ball M, Bartlett M, Sinclair A. Lipoprotein(a), essential fatty acid status and lipoprotein lipids in female Australian vegetarians. Clin Sci 1999; 97: 175-181.
9. Li D, Sinclair A, Mann N, Turner A, Ball M, Kelly F, Abedin L, Wilson A. The association of diet and thrombotic risk factors in healthy male vegetarians and meat-eaters. Eur J Clin Nutr 1999; 53: 612-619.
10. Ashton YM, Mann NJ. The contribution of food groups to the total fat and fat type in various diets. Proc Nutr Soc Aust 2000; 24: 104.
11. McLennan W, Podger A. National Nutrition Survey, Selected Highlights, Australia. Canberra: AGPS, 1997.
12. Keenan JM and Morris DH. Hypercholesterolemia. Dietary advice for patients regarding meat. Postgrad Med 1995; 98: 113-114, 117-118, 120-121 passim.
13. http://www.heartfoundation.com.au
14. Sinclair AJ, Slattery WJ, O'Dea K. The analyses of polyunsaturated fatty acids in meat by capillary gas liquid chromatography. J Sci Food Agric 1982; 33: 771-776.
15. Sinclair AJ, O'Dea K. The lipid levels and fatty acid compositions of the lean portions of Australian beef and lamb. Food Technol Aust 1987; 35: 228-231.
16. Hornstein L, Crowe PF, Hiner R. Composition of lipids in some beef muscles. J Food Sci 1967; 32: 650-655.
17. McDonald IW, Scott TW. Foods of ruminant origin with elevated content of polyunsaturated fatty acids. World Rev Nutr Diet 1977; 26: 144-207.
18. Li D, Ng A, Mann NJ, Sinclair AJ. Contribution of meat fat to dietary arachidonic acid. Lipids. 1998; 33: 437-440.
19. Sinclair AJ, O'Dea K. The lipid levels and fatty acid composition of the lean portions of pork, chicken and rabbit meats. Food Technol Aust 1987; 35: 232-240.
20. Mann NJ, Johnson LG, Warrick GE, Sinclair AJ. The arachidonic acid content of the Australian diet is lower than previously estimated. J Nutr 1995; 125: 2528-2535.
21. O'Dea K, Trainanedes K, Chisolm K, Leyden H, Sinclair AJ. Cholesterol-lowering effect of a low fat diet containing lean beef is reversed by the addition of beef fat. Am J Clin Nutr 1990; 52: 491-494.
22. Morgan S, O'Dea K, Sinclair AJ. Low-fat diets rich in lean beef: The effects of the addition of safflower and olive oil. J Amer Diet Assoc 1993; 93: 644-648.
23. Morgan SA, O'Dea K, Sinclair AJ. A low-fat diet supplemented with monounsaturated fat results in less HDL-C lowering than a very-low-fat diet. J Am Diet Assoc 1997; 97: 151-156.
24. Watts GF, Ahmed W, Quiney J, Houlston R, Jackson P, Iles C, Lewis B. Effective lipid lowering diets including lean meat. Br Med J 1988; 296: 235-237.
25 Beauchesne-Rondeau E, Gascon A, Bergeron J, Jacques H. Plasma lipids and lipoproteins in hypercholesterolemic men fed a lipid-lowering diet containing lean beef, lean fish, or poultry. Am J Clin Nutr 2003; 77: 587-593.
26. Scott LW, Dunn JK, Pownall HJ, Brauchi DJ, McMann MC, Herd JA, Harris KB, Savell JW, Cross HR, Gotto AM Jr. Effects of beef and chicken consumption on plasma lipid levels in hypercholesterolemic men. Arch Intern Med 1994; 154: 1261-1267.
27. Davidson MH, Hunninghake D, Maki KC, Kwiterovich PO Jr, Kafonek S. Comparison of the effects of lean red meat vs lean white meat on serum lipid levels among free-living persons with hypercholesterolemia: a long-term, randomized clinical trial. Arch Intern Med 1999; 159: 1331-1338.
28. Anderson JW, Johnstone BM, Cook-Newell ME. Meta-analysis of the effects of soy protein intake on serum lipids. N Engl J Med 1995; 333: 276-282.
29. Ho SC, Woo JL, Leung SS, Sham AL, Lam TH, Janus ED. Intake of soy products is associated with better plasma lipid profiles in the Hong Kong Chinese population. J Nutr 2000; 130: 2590-2593.
30. Yamashita T, Sasahara T, Pomeroy SE, Collier G, Nestel PJ. Arterial compliance, blood pressure, plasma leptin, and plasma lipids in women are improved with weight reduction equally with a meat-based diet and a plant-based diet. Metabolism 1998; 47: 1308-1314.
31. Ashton E, Ball M. Effects of soy as tofu vs meat on lipoprotein concentrations. Eur J Clin Nutr 2000; 54: 14-19.
32. Hamberg M, Svensson J, Samuelsson B. Thromboxanes: a new group of biologically active compounds derived from prostagladin endoperoxides. Proc Nat Acad Sci 1975; 72: 2994 - 2998.
33. Moncada S, Vane JR. Arachidonic acid metabolites and the interactions between platelets and blood vessel walls. N Engl J Med 1979; 300: 1142-1148.
34. Sinclair AJ, O'Dea K, Dunstan G, Ireland PD, Niall M. The effects of plasma lipids and fatty acid composition of very low fat diets enriched with fish or kangaroo meat. Lipids 1987; 22: 523-529.
35. Sinclair AJ, Johnson L, O'Dea K, Holman RT. Diets rich in lean beef increase the eicosatrienoic, arachidonic, eicosapentaenoic and docosapentaenoic acid content of plasma phospholipids. Lipids 1994; 29: 337-343.
36. Mann N, Sinclair A, Pille M, Johnson L, Warrick G, Reder E, Lorenz R. The effect of short-term diets rich in fish, red meat, or white meat on thromboxane and prostacyclin synthesis in humans. Lipids 1997; 32: 635-644.
37. Hornstra G. Dietary fats, prostanoids and arterial thrombosis. Monograph. Martinus Nijhoff Publishers. The Hague, 1982.

38. Hornstra G. Dietary lipids, platelet function and arterial thrombosis in animals and man. Proc Nutr Soc 1985; 44: 371-378.
39. Mitropoulos KA, Reeves BE, Miller GJ. The activation of factor VII in citrated plasma by charged long-chain saturated fatty acids at the interface of large triglyceride-rich lipoproteins. Blood Coagul Fibrinolysis 1993; 4: 943-51.
40. Mitropoulos KA, Miller GJ, Howarth DJ, Reeves BE, Cooper JA. The effects of intravenous Triton WR-1339 on factor VII coagulant activity and plasma lipoproteins in normocholesterolaemic and hypercholesterolaemic rabbits. Blood Coagul Fibrinolysis 1994; 5: 583-91.
41. Dougherty RM, Allman MA, Iacano JM. Effects of diets containing high or low amounts of stearic acid on plasma lipoprotein fractions and fecal fatty acid excretion of men. Am J Clin Nutr 1995; 61: 1120-1128.
42. Kelly FD, Sinclair AJ, Mann NJ, Turner AH, Abedin L, Li D. A stearic acid-rich diet improves thrombogenic and atherogenic risk factor profiles in healthy males. Eur J Clin Nutr 2001; 55: 88-96.
43. Ashton EL, Dalais FS, Ball MJ. Effect of meat replacement by tofu on CHD risk factors including copper induced LDL oxidation. J Am Coll Nutr 2000; 19: 761-767.
44. Cleland LG, James MJ, Neumann MA, D'Angelo M, Gibson RA. Linoleate inhibits EPA incorporation from dietary fish-oil supplements in human subjects. Am J Clin Nutr 1992; 55: 395-399.
45. Li D, Zhang H,Hsu-Hage BHH, Wahlqvist ML, Sinclair AJ. The influence of fish, meat and polyunsaturated fat intakes on platelet phospholipid polyunsaturated fatty acids in male Melbourne Chinese and Caucasian. Eur J Clin Nutr 2001; 55: 1036-1042.
46. Li D, Sinclair A, Wilson A, Nakkote S, Kelly F, Abedin L, Mann N, Turner A. Effect of dietary alpha-linolenic acid on thrombotic risk factors in vegetarian men. Am J Clin Nutr 1999; 69: 872-882.
47. Meyer BJ, Howe PRC, Lewis J, Milligan G, Mann NJ, Sinclair AJ. Australian intakes and food sources of omega-6 and omega-3 polyunsaturated fatty acids. Proc Nutr Soc Aust 2000; 24: 96.
48. Arnesen E, Refsum H, Bonaa KH, Ueland PM, Forde OH, Nordrehaug JE. Serum total homocysteine and coronary heart disease. Int J Epidemiol 1995; 24: 704-9.
49. Verhoef P, Stampfer MJ, Buring JE, Gaziano JM, Allen RH, Stabler SP, Reynolds RD, Kok FJ, Hennekens CH, Willett WC. Homocysteine metabolism and risk of myocardial infarction: Relation with vitamins B6 and B12 and folate. Am J Epidem 1996; 143: 845-859.
50. Nicklas TA, Farris RP, Myers L, Berenson GS. Impact of meat consumption on nutritional quality and cardiovascular risk factors in young adults: the Bogalusa Heart Study. J Am Diet Assoc 1995; 95: 887-892.
51. Alexander D, Ball MJ, Mann J. Nutrient intake and haematological status of vegetarians and age-sex matched omnivores. Eur J Clin Nutr 1994; 48: 538-46.
52. Li D, Sinclair A, Mann N, Turner A, Ball M. Selected micronutrient intake and status in men with differing meat intakes, vegetarians and vegans. Asia Pac J Clin Nutr 2000; 9: 18-23.
53. Boushey C, Beresford S, Omenn G, Motulsky A. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. Probable benefits of increasing folic acid intakes. JAMA 1995; 274: 1049-1057.
54. Mann NJ, Li D, Sinclair AJ, Dudman NP, Guo XW, Elsworth GR, Wilson AK, Kelly FD. The effect of diet on plasma homocysteine concentrations in healthy male subjects. Eur J Clin Nutr 1999; 53: 895-9.